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ORIGINAL RESEARCH

Development and Validation of Nomogram for the Prediction of Malignant Ventricular Arrhythmia Including Circulating Inflammatory Cells in Patients with Acute ST-Segment Elevation Myocardial Infarction

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Background: Malignant ventricular arrhythmia (MVA) can seriously affect the hemodynamic changes of the body. In this study, we developed and validated a nomogram to predict the in-hospital MVA risk in patients with STEMI after emergency PCI.

Methods: The multivariable logistic regression analysis included variables with a P<0.05 in the univariate logistic regression analysis and investigated the independent predictors affecting in-hospital MVA after PCI in patients with STEMI in the training cohort. The construction of a nomogram model used independent predictors to predict the risk of in-hospital MVA, and C-index, Hosmer-Lemeshow (HL) test, calibration curves, decision curve analysis (DCA), and receiver operating characteristic (ROC) were used to validate the nomogram.

Results: Killip class [OR=5.034 (95% CI: 1.596–15.809), P=0.005], CK-MB [OR=1.002 (95% CI: 1.001–1.004), P=0.022], serum potassium [OR=0.618 (95% CI: 0.406–0.918), P=0.020], NLR [OR=1.073 (95% CI: 1.034–1.115), P<0.001], and monocyte [OR=1.974 (95% CI: 1.376–2.925), P<0.001] were the independent predictors of in-hospital MVA after PCI in patients with STEMI. A nomogram including the 5 independent predictors was developed to predict the risk of in-hospital MVA. The C-index, equivalent to the area under the ROC curve (AUC), was 0.803 (95% confidence interval [CI]: 0.738–0.868) in the training cohort, and 0.801 (95% CI:0.692–0.911) in the validation cohort, showing that the nomogram had a good discrimination. The HL test (χ^2=8.439, P=0.392 in the training cohort; χ^2=9.730, P=0.285 in the validation cohort) revealed a good calibration. The DCA suggested an obvious clinical net benefit.

Conclusion: Killip class, CK-MB, serum potassium, NLR, and monocyte were independent factors for in-hospital MVA after PCI in patients with STEMI. The nomogram model constructed based on the above factors to predict the risk of in-hospital MVA had satisfactory discrimination, calibration, and clinical effectiveness, and was an excellent tool for early prediction of the risk of in-hospital MVA after PCI in patients with STEMI.

Keywords: ST-segment elevation myocardial infarction, percutaneous coronary intervention, nomogram model, malignant ventricular arrhythmia, circulating inflammatory cells

Introduction

Ischemic heart disease is a critical cause of death in China, resulting in a large number of deaths and disabilities every year.[1] ST-segment elevation myocardial infarction (STEMI) is a serious condition of ischemic heart disease with high mortality and hospitalization rates.[2] Currently, percutaneous coronary intervention (PCI) is one of the most reliable methods for treating STEMI.[3] PCI allows timely reperfusion of the infarcted vessel, but reperfusion itself can exacerbate myocardial injury and increase the likelihood of malignant ventricular arrhythmia (MVA). As a fatal complication after STEMI, MVA can seriously affect the hemodynamic changes of the body, leading to syncope and even sudden cardiac death.[4] It has been shown that the majority of MVA events occur within 48 hours of the onset of STEMI symptoms and become one of the most common causes

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of sudden cardiac death in patients during hospitalization.[5,6] Compared with patients without MVA, patients with MVA have a significantly lower 30-day survival rate (69% vs 96%).[7] However, death caused by MVA is often regarded as the first symptom of STEMI, as it is usually sudden and unexpected with no obvious clinical indications, which lead to a possible underestimation of the true burden of MVA.[8] Therefore, identifying high-risk STEMI patients with MVA early in admission and taking effective preventive management measures are crucial for improving the survival of these patients. The development of a convenient and effective prediction model is useful for evaluating the risk of in-hospital MVA after emergency PCI in patients with STEMI and is important for early detection of patients at high risk of MVA and timely intervention.

The development of MVA is strongly associated with multiple patient characteristics including low blood pressure, low left ventricular ejection fraction (LVEF), and hypokalemia, creatine kinase-MB (CK-MB), cardiac troponin I (cTnI), creatinine, hyperlipidaemia.[9–15] In addition, previous studies have shown that increased circulating inflammatory cells, such as macrophages, neutrophils, and monocytes, are associated with the development of arrhythmia in patients with ischemia.[16–18] Moreover, circulating inflammatory cell tests, including lymphocytes, monocytes, and neutrophils, are easy to conduct, inexpensive, and present information about cell types and morphological parameters. It indicates that circulating inflammatory cells may provide useful information for predicting the risk of MVA in patients with STEMI. Based on the above information, we speculate that the occurrence of MVA in patients with STEMI could be better predicted by the combination of patient characteristics and circulating inflammatory cell.

The nomogram is a model that allows for the prediction of individual disease risk, with graphical and visual features, and easy to be applied in clinical practice. In this study, we analyzed the patient characteristics and circulating inflammatory cells of patients with STEMI before emergency PCI, and developed and validated a nomogram to predict the in-hospital MVA risk in patients with STEMI after emergency PCI, helping clinicians assess the patient's condition.

Methods

Ethics Statement

This retrospective study was approved by medical ethics committee of Shuyang Hospital of Traditional Chinese Medicine (No. 202303) and conducted in accordance with the Declaration of Helsinki. Written informed consents were obtained from patients.

Study Population

A retrospective analysis of 936 patients with STEMI at Shuyang Hospital of Traditional Chinese Medicine from February 2019 to February 2022 was performed. Inclusion criteria (1) age≥18 years; (2) eligible for the diagnostic criteria of STEMI; (3) emergency PCI within first 24 h from symptom onset. Exclusion criteria: (1) with malignant tumor, active infection, severe major organ dysfunction; (2) treated with steroid therapy for autoimmune disease; (3) loss of research information. After screening according to the inclusion and exclusion criteria, 456 patients were included in this study. Three hundred and nineteen patients enrolled between February 2019 and March 2021 were assigned to the training cohort and 137 patients enrolled between April 2021 and February 2022 were assigned to the validation cohort (Figure 1). All patients were divided into the MVA group (n=76) and the non-MVA group (n=380) based on whether MVA happened in the hospital after PCI. Referring to the rule to have at least 10 outcome events per variable,[19,20] we ensured less than 8 features retained for the model in order to avoid underpowered. MVA was defined as ventricular tachycardia (VT) and ventricular fibrillation (VF).

Data Collection

Patient characteristics and circulating inflammatory cells of patients with STEMI at admission were collected, including age, gender, body mass index (BMI), systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate, killip class, left ventricular ejection fraction (LVEF), anterior wall myocardial infarction (MI), history of MI, history of hypertension, history of coronary heart disease, history of angina pectoris, history of diabetes mellitus, history of hyperlipidaemia, history of cerebrovascular disease, history of smoking, CK-MB, cTnI, serum potassium, creatinine, white blood cell (WBC), neutrophil, lymphocyte, neutrophil-to-lymphocyte ratio (NLR), monocyte, eosinophil, basophil. Moreover, MVA data during hospitalization were collected.

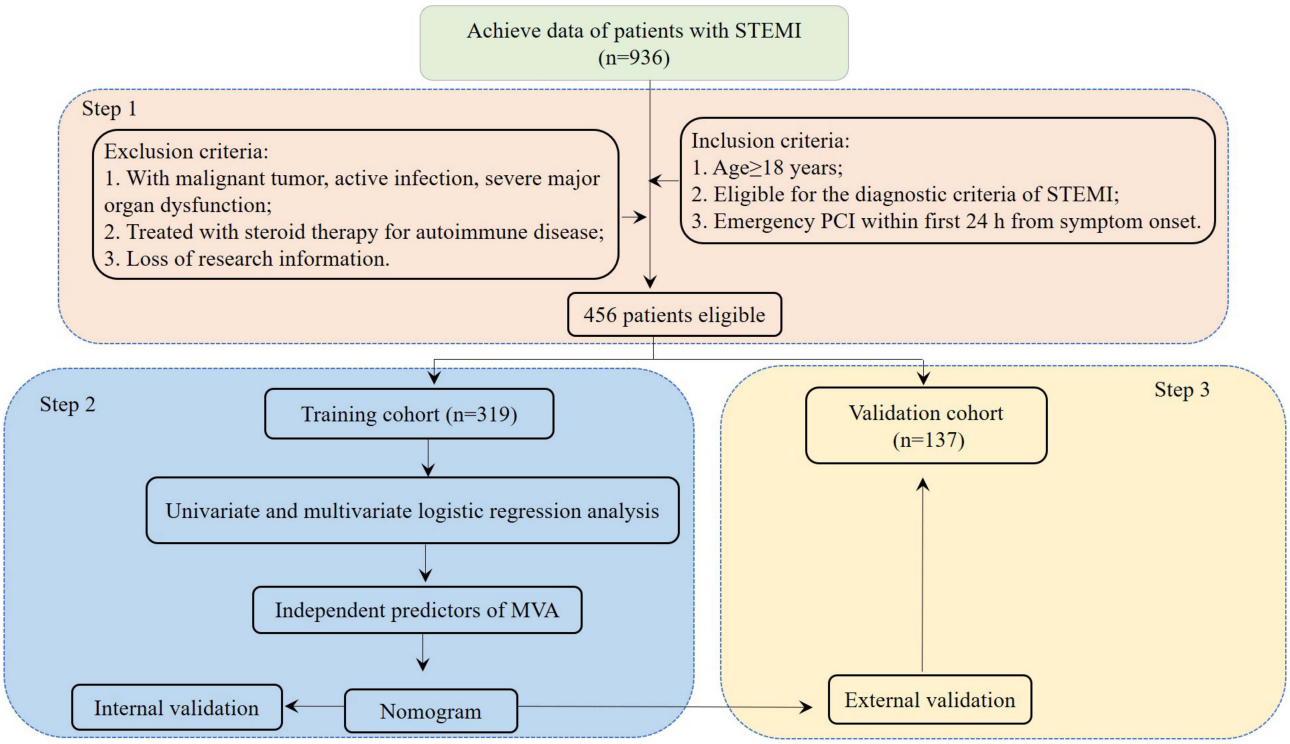

Figure 1 Summary of patient flow diagram. Step 1: patient screening; Step 2: model construction; Step 3: model validation.
Abbreviations: STEMI, ST-segment elevation myocardial infarction; PCI, percutaneous coronary intervention; MVA, malignant ventricular arrhythmia.

Nomogram Construction and Validation

Following the univariate logistic regression analysis, the multivariable logistic regression analysis included variables with a P<0.05 in the univariate logistic regression analysis and investigated the independent predictors affecting in-hospital MVA after PCI in patients with STEMI in the training cohort. The construction of a nomogram model using independent predictors to predict the risk of in-hospital MVA after PCI in patients with STEMI, and C-index was applied to evaluate the predictive efficacy of the nomogram model. Bootstrap multiple sampling 1000 times was applied for the validation of the nomogram in the training and validation cohorts, respectively. The Hosmer-Lemeshow (HL) test assessed the model fit, and the calibration of the model was evaluated by plotting calibration curves. Decision curve analysis (DCA) was drawn to assess the net benefit rate of this model. Receiver operating characteristic (ROC) curves were drawn to assess the discrimination of the model.

Statistical Methods

Kolmogorov–Smirnov normality test was performed on the continuous variables. The skewed distribution was expressed as median (inter-quartile range [IQR]), and Mann–Whitney *U*-test was applied to compare the two groups. The categorical data was expressed as frequency (percentage), and chi-square test was applied to compare the two groups. All statistical analyses were conducted using two-sided tests, and P <0.05 was regarded as statistically significant. The statistical analyses were performed using SPSS 23.0 and R4.2.1.

Results

Study Cohort

A total of 936 patients underwent screening. There were 456 patients with STEMI enrolled, including 76 (16.7%) patients in the MVA group. A total of 319 patients were chosen for the training cohort, and 137 patients for the validation cohort (Figure 1). The patient characteristics and circulating inflammatory cells of the training and validation cohorts were presented in Table 1. No statistically significant differences were observed between the two cohorts in terms of the patient characteristics and circulating inflammatory cells (all P values >0.05).

Table 1 Baseline Characteristics of Patients in the Training and Validation Cohorts

Variables	Training Cohort (n=319)	Validation Cohort (n=137)	P value
Patient characteristics			
Age, years, median (IQR)	60.00 (54.00, 64.00)	61.00 (56.00, 64.00)	0.384#
Gender, n (%)			0.893*
Male	248 (77.7)	108 (78.8)	
Female	71 (22.3)	29 (21.2)	
BMI, kg/m^2, median (IQR)	23.94 (22.87, 25.27)	24.20 (22.98, 25.32)	0.374#
SBP, mmHg, median (IQR)	118.00 (105.00, 127.00)	116.00 (111.00, 126.00)	0.506#
DBP, mmHg, median (IQR)	79.00 (67.00, 89.00)	80.00 (69.00, 90.00)	0.624#
Heart rate, beats/min, median (IQR)	80.00 (73.00, 87.00)	80.00 (71.00, 87.00)	0.984#
Killip class, n (%)			0.983*
I	214 (67.1)	91 (66.4)	
II	64 (20.1)	27 (19.7)	
III	21 (6.6)	9 (6.6)	
IV	20 (6.3)	10 (7.3)	
LVEF, %, median (IQR)	47.00 (45.00, 51.00)	47.00 (44.00, 50.00)	0.551#
Anterior wall MI, n (%)			0.934*
Yes	166 (52.0)	70 (51.1)	
No	153 (48.0)	67 (48.9)	
History of MI, n (%)			0.891*
Yes	19 (6.0)	7 (5.1)	
No	300 (94.0)	130 (94.9)	
History of hypertension, n (%)			0.983*
Yes	154 (48.3)	67 (48.9)	
No	165 (51.7)	70 (51.1)	
History of coronary heart disease, n (%)			0.759*
Yes	35 (11.0)	13 (9.5)	
No	284 (89.0)	124 (90.5)	
History of angina pectoris, n (%)			0.896*
Yes	78 (24.5)	32 (23.4)	
No	241 (75.5)	105 (76.6)	
History of diabetes mellitus, n (%)			0.993*
Yes	46 (14.4)	19 (13.9)	
No	273 (85.6)	118 (86.1)	

(*Continued*)

Table 1 (Continued).

Variables	Training Cohort (n=319)	Validation Cohort (n=137)	P value
History of hyperlipidaemia, n (%)			0.887*
Yes	153 (48.0)	64 (46.7)	
No	166 (52.0)	73 (53.3)	
History of cerebrovascular disease, n (%)			0.847*
Yes	41 (12.9)	16 (11.7)	
No	278 (87.1)	121 (88.3)	
History of smoking, n (%)			0.836*
Yes	144 (45.1)	64 (46.7)	
No	175 (54.9)	73 (53.3)	
CK-MB, U/L, median (IQR)	111.94 (67.63, 191.39)	104.99 (50.16, 196.57)	0.498#
cTnI, ng/mL, median (IQR)	33.90 (21.40, 47.80)	30.60 (18.50, 45.40)	0.193#
Serum potassium, mmol/L, median (IQR)	3.67 (2.74, 3.86)	3.50 (2.72, 4.27)	0.785#
Creatinine, umol/L, median (IQR)	64.90 (59.45, 70.90)	65.60 (58.30, 70.40)	0.642#
Circulating inflammatory cells			
WBC, $\times 10^9$/L, median (IQR)	10.18 (9.05, 11.20)	10.10 (8.63, 11.26)	0.300#
Neutrophil, $\times 10^9$/L, median (IQR)	8.34 (6.51, 9.55)	8.10 (7.03, 10.39)	0.304#
Lymphocyte, $\times 10^9$/L, median (IQR)	2.13 (1.42, 4.02)	1.89 (1.13, 3.79)	0.120#
NLR, %, median (IQR)	3.90 (1.95, 6.40)	4.50 (2.20, 6.80)	0.138#
Monocyte, $\times 10^9$/L, median (IQR)	0.74 (0.42, 1.16)	0.64 (0.40, 1.09)	0.290#
Eosinophil, $\times 10^9$/L, median (IQR)	0.32 (0.17, 0.46)	0.32 (0.19, 0.49)	0.443#
Basophil, $\times 10^9$/L, median (IQR)	0.08 (0.05, 0.12)	0.09 (0.05, 0.13)	0.212#

Notes: *For chi-square test; #For Mann–Whitney *U*-test.
Abbreviations: IQR, inter-quartile range; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; LVEF, left ventricular ejection fraction; MI, myocardial infarction; CK-MB, creatine kinase-MB; cTnI, cardiac troponin I; WBC, white blood cell; NLR, neutrophil-to-lymphocyte ratio.

Independent Predictors for MVA

There were 7 feature variables (SBP, Killip class, LVEF, CK-MB, serum potassium, NLR, and monocyte) selected by the univariate logistic regression analysis, which were associated with in-hospital MVA after PCI in patients with STEMI (Table 2). The multivariate analysis showed that 5 feature variables, namely Killip class [OR=5.034 (95% CI: 1.596–15.809), P=0.005], CK-MB [OR=1.002 (95% CI: 1.001–1.004), P=0.022], serum potassium [OR=0.618 (95% CI: 0.406–0.918), P=0.020], NLR [OR=1.073 (95% CI: 1.034–1.115), P<0.001], and monocyte [OR=1.974 (95% CI: 1.376–2.925), P<0.001], were the independent predictors of in-hospital MVA after PCI in patients with STEMI (Table 2).

Nomogram Establishment

A nomogram including the 5 independent predictors (Killip class, CK-MB, serum potassium, NLR, and monocyte) was developed to predict the risk of in-hospital MVA after PCI in patients with STEMI (Figure 2). Each predictor corresponds to a given score on the point axis of the nomogram, and the scores of 5 predictors are summed to achieve the total points.

Table 2 Univariate and Multivariate Logistic Analyses to Determine the Independent Predictors Associated with Malignant Ventricular Arrhythmia in the Training Cohort

Variables	Univariate Logistic Regression			Multivariable Logistic Regression		
	P value	OR	95% CI	P value	OR	95% CI
Patient characteristics						
Age, years	0.636	1.01	0.970–1.052			
Gender (male vs female)	0.664	0.858	0.440–1.765			
BMI, kg/m2	0.151	1.098	0.966–1.249			
SBP, mmHg	0.03	0.979	0.960–0.998	0.218	0.985	0.961–1.009
DBP, mmHg	0.244	0.987	0.966–1.009			
Heart rate, beats/min	0.285	1.016	0.986–1.047			
Killip class, n (%)						
I	Reference			Reference		
II	0.352	1.437	0.646–3.022	0.230	1.681	0.698–3.863
III	0.162	2.164	0.665–6.050	0.146	2.445	0.669–7.760
IV	<0.001	6.926	2.619–18.439	0.005	5.034	1.596–15.809
LVEF, %	0.003	0.942	0.904–0.980	0.139	0.965	0.920–1.010
Anterior wall MI (yes vs no)	0.669	1.138	0.630–2.070			
History of MI (yes vs no)	0.593	1.366	0.377–3.953			
History of hypertension (yes vs no)	0.437	0.79	0.433–1.427			
History of coronary heart disease (yes vs no)	0.929	1.043	0.374–2.498			
History of angina pectoris (yes vs no)	0.476	1.272	0.641–2.424			
History of diabetes mellitus (yes vs no)	0.783	0.886	0.345–1.995			
History of hyperlipidaemia (yes vs no)	0.861	1.054	0.582–1.904			
History of cerebrovascular disease (yes vs no)	0.594	1.255	0.512–2.779			
History of smoking (yes vs no)	0.745	1.103	0.608–1.992			
CK-MB, U/L	0.044	1.002	1.000–1.003	0.022	1.002	1.001–1.004
cTnI, ng/mL	0.961	1	0.983–1.016			
Serum potassium, mmol/L	0.029	0.678	0.474–0.956	0.020	0.618	0.406–0.918
Creatinine, umol/L	0.123	1.003	0.999–1.007			
Circulating inflammatory cells						
WBC, $\times 10^9$/L	0.099	0.869	0.735–1.028			
Neutrophil, $\times 10^9$/L	0.554	0.983	0.921–1.035			
Lymphocyte, $\times 10^9$/L	0.297	1.05	0.952–1.149			

(*Continued*)

Table 2 (Continued).

Variables	Univariate Logistic Regression			Multivariable Logistic Regression		
	P value	OR	95% CI	P value	OR	95% CI
NLR, %	<0.001	1.084	1.048–1.123	<0.001	1.073	1.034–1.115
Monocyte, $\times 10^9$/L	<0.001	1.969	1.421–2.804	<0.001	1.974	1.376–2.925
Eosinophil, $\times 10^9$/L	0.593	1.156	0.627–1.896			
Basophil, $\times 10^9$/L	0.102	6.119	0.597–52.055			

Abbreviations: BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; LVEF, left ventricular ejection fraction; MI, myocardial infarction; CK-MB, creatine kinase-MB; cTnI, cardiac troponin I; WBC, white blood cells; NLR, neutrophil-to-lymphocyte ratio; OR, odds ratio; CI, confidence interval.

The total points corresponding to the probability of MVA at the bottom of the nomogram shows that patients with higher total points are more likely to experience MVA during hospitalization.

Nomogram Verification

The C-index, equivalent to the area under the ROC curve (AUC), was 0.803 (95% confidence interval [CI]: 0.738–0.868) in the training cohort, and 0.801 (95% CI:0.692–0.911) in the validation cohort, showing that the nomogram had a good discrimination. The ROC curves of this model in the training and validation cohorts were plotted in Figures 3A and D, respectively. The positive and negative perfective value was 68·8% and 91·8% in the training cohort, respectively. The HL test (χ^2=8.439, P=0.392 in the training cohort; χ^2=9.730, P=0.285 in the validation cohort) revealed a good calibration. The calibration curves of this nomogram in both cohorts were plotted in Figures 3B and E. The DCA suggested an obvious clinical net benefit in the training cohort (Figure 3C) and in the validation cohort (Figure 3F). If circulating inflammatory cells was excluded, the C-index of the nomogram was 0.721 (95% CI: 0.647–0.795), which was lower than the model with circulating inflammatory cells (Figure 4).

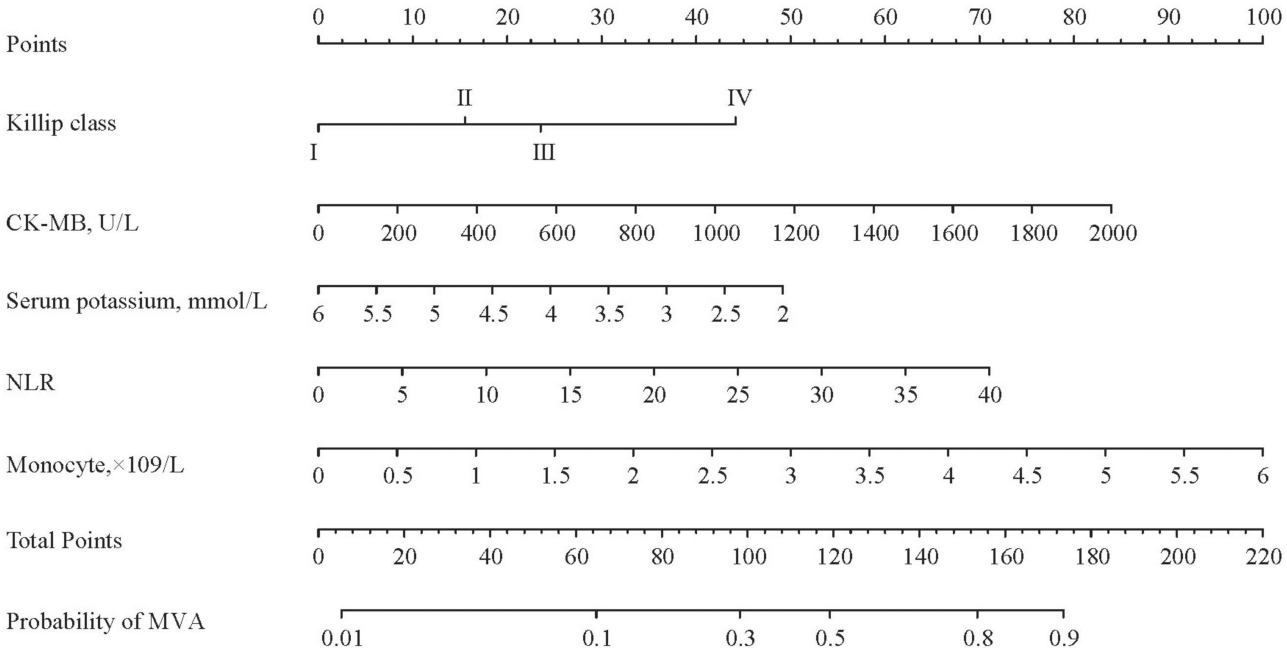

Figure 2 Nomogram for predicting malignant ventricular arrhythmia in patients with ST- segment elevation myocardial infarction.
Abbreviations: CK-MB, creatine kinase-MB; NLR, neutrophil-lymphocyte ratio; MVA, malignant ventricular arrhythmia.

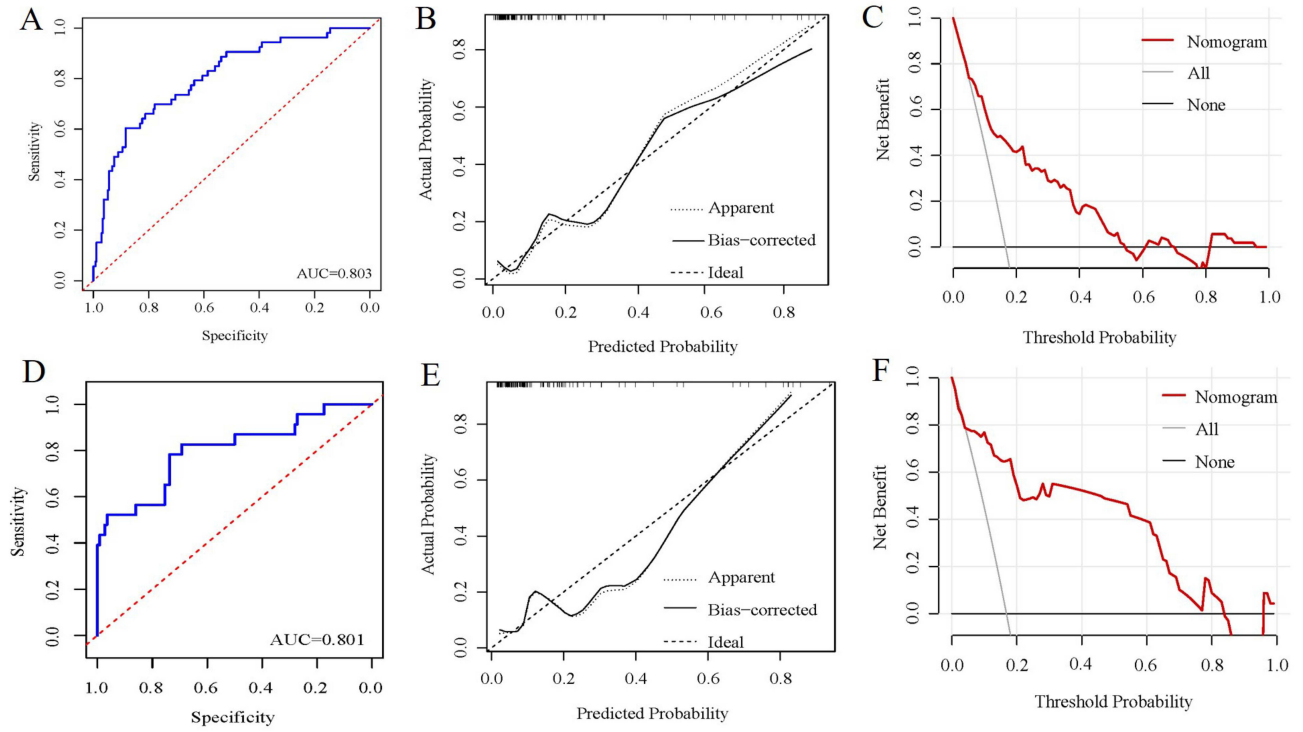

Figure 3 Nomogram verification using training and validation cohorts [(**A**–**C**) for training cohort and (**D**–**F**) for validation cohort]. The AUCs of ROC curves are 0.803 (**A**) and 0.801 (**D**); Both calibration plots (**B** and **E**) show good agreement. The DCA plots (**C** and **F**) suggest that the risk of in-hospital MVA predicted by the nomogram yield net benefit in both cohorts.

Abbreviations: AUC, areas under the curve; ROC, receiver operating characteristic; DCA, decision curve analysis; MVA, malignant ventricular arrhythmia.

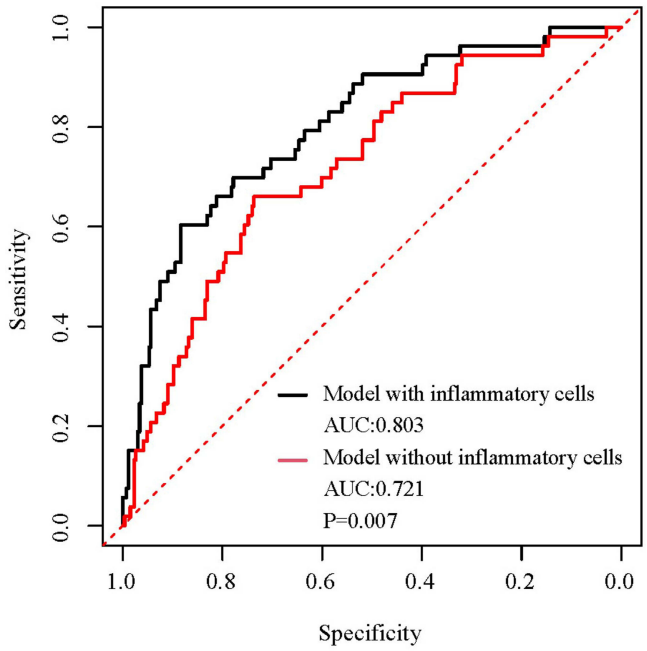

Figure 4 The AUC (C-index) of the nomogram with circulating inflammatory cells was 0.803 (95% CI: 0.738–0.868), demonstrating very good discrimination. The AUC (C-index) of the nomogram without circulating inflammatory cells was 0.721 (95% CI: 0.647–0.795).

Abbreviations: 95% CI, 95% confidence interval; AUC, areas under the curve.

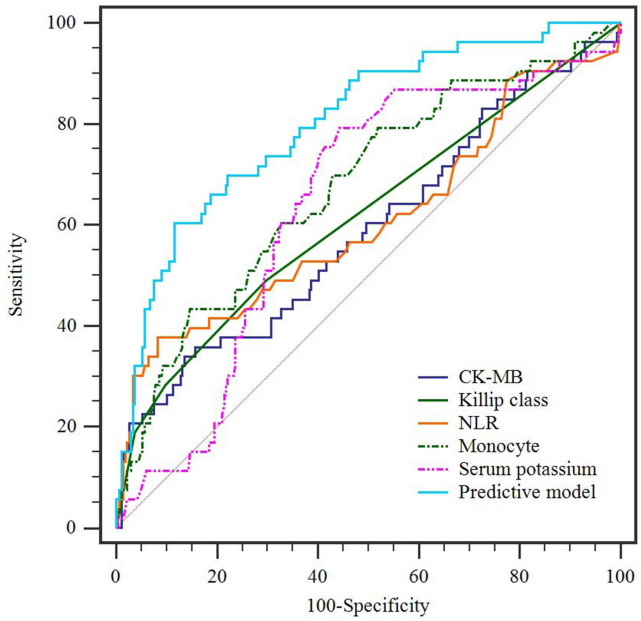

Figure 5 Comparison for the ROC curves generated based on each independent factor and predictive model.
Abbreviations: ROC, receiver operating characteristic; CK-MB, creatine kinase-MB; NLR, neutrophil-lymphocyte ratio.

Predictive Performances of Each Independent Predictor and Predictive Model

Combined application of Killip class, CK-MB, serum potassium, NLR, and monocyte showed the highest AUC (AUC: 0.803, 95% CI: 0.738–0.868), which revealed that the predictive model improved the predictive power compared with the single predictors (Killip class [AUC: 0.619, 95% CI: 0.563–0.672], CK-MB [AUC: 0.589, 95% CI: 0.533–0.644], serum potassium [AUC: 0.641, 95% CI: 0.586–0.694], NLR [AUC: 0.607, 95% CI: 0.552–0.661], and monocyte [AUC: 0.677, 95% CI: 0.623–0.728]) (Figure 5).

Discussion

Although PCI can quickly restore myocardial perfusion and improve the prognosis of patients with STEMI, the risk of MVA remains high in patients after PCI. MVA is one of the serious complications in patients with STEMI and the main cause of early death.[21] Therefore, the early identification of STEMI patients at high risk of developing MVA is an urgent need. In this study, we aimed to develop and validate a nomogram for the prediction of MVA based on patient characteristics and circulating inflammatory cells in patients with acute STEMI, helping clinicians detect MVA early.

Killip classification is used to evaluate the degree of heart failure after acute myocardial infarction.[22,23] It has been reported that myocardial infarction patients with higher Killip class often suffer from worse coronary artery disease and larger myocardial infarction area, which implies more myocardial necrosis and subsequent replacement of the necrosis by fibrotic scars, which is hard to reverse once developed, apart from the impact on cardiac contractility, which disrupts the normal cardiac electrical activity and results in arrhythmia, which may be a reason for the poor outcome of patients with a higher Killip class.[24,25] Nienhuis et al[26] reported that CK-MB is also a useful predictor of the degree of myocardial injury and prognosis in patients with MI. In addition, it is widely accepted that the early onset of MI is usually accompanied by electrolyte disturbances, especially hypokalemia. Serum potassium plays an important role in maintaining the normal activity of myocardial cells. Hypokalemia can cause abnormal membrane potential of myocardial cells, which makes patients prone to potentially fatal arrhythmia.[27–30] In the present study, STEMI patients with MVA had a higher Killip class (OR=5.034, P=0.005), higher CK-MB levels (OR=1.002, P=0.022), and lower serum potassium levels (OR=0.618, P=0.020) compared with those without MVA, which is consistent with previous studies.

In addition to the killip classification, CK-MB, and serum potassium, the NLR and monocyte were observed to be the independent predictors of MVA in patients with STEMI in this study. Inflammatory response, as one of the pathogenesis

of atherosclerosis, is an important risk factor for cardiovascular events.[31,32] NLR, the neutrophil-to-lymphocyte ratio, can compensate for mutual deficiencies and provides a more stable and flexible response to the inflammatory status of the body.[33] It has been reported that the increased NLR is an independent predictor of poor prognosis after PCI in patients with MI.[34,35] van der Laan et al[36] reported that high levels of monocytes in patients with STEMI were significantly associated with extensive myocardial injury, as shown by large infarct size, increased transmural infarct extent, and the existence of microvascular obstruction at baseline. Tsujioka et al assayed the levels of monocyte in 36 patients with STEMI and observed that higher level of monocytes was associated with impaired recovery of left ventricular function.[37]

The main strength of our study is the development of a visual nomogram including circulating inflammatory cells with good predictive accuracy for the early identification of MVA in patients with STEMI. It supports individualized decision for MVA risk assessment and was superior to the nomogram developed without circulating inflammatory cells. The discrimination and calibration of the nomogram including circulating inflammatory cells were verified to be good and the model also yielded net benefits in both the training and validation cohorts. Compared with previous study reported by Zheng et al[38] that predicted the risk of in-hospital complicating ventricular tachyarrhythmia after acute myocardial infarction, our study provided some new information by using circulating inflammatory cells in the present model. The circulating inflammatory cell counts are easy to carry out and inexpensive. Moreover, the C-index of the present nomogram was 0.803, which was higher than that in the study of Zheng et al (C-index 0.764).[38]

There were 2 limitations in this study. Firstly, this study was a retrospective design and included patients with complete clinical data, leading to a selection bias. Secondly, because this study was single-center and had a limited sample size, which may limit its generalization. Therefore, a large-sample, multicenter, prospective study is needed to further explore the factors influencing in-hospital MVA after PCI in patients with STEMI and improve the nomogram model.

In conclusion, Killip class, CK-MB, serum potassium, NLR, and monocyte are independent factors for in-hospital MVA after PCI in patients with STEMI. The nomogram model constructed based on the above factors to predict the risk of in-hospital MVA after PCI in patients with STEMI has satisfactory discrimination, calibration, and clinical effectiveness, and is an excellent tool for early prediction of the risk of in-hospital MVA after PCI in patients with STEMI. Our findings allow clinicians assess the patient's condition and intervene proactively before in-hospital MVA occurs.

Data Sharing Statement

All data generated or analysed during this study are included in this article. Further enquiries can be directed to the corresponding author.

Ethical Approval

This retrospective study was approved by medical ethics committee of Shuyang Hospital of Traditional Chinese Medicine (No. 202303) and conducted in accordance with the Declaration of Helsinki.

Informed Consent

Data collection was performed after written informed consents were obtained from subjects.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors have no conflicts of interest to declare.

References

1. Hao Y, Zhao D, Liu J, et al. Performance of management strategies with class I recommendations among patients hospitalized with ST-segment elevation myocardial infarction in China. *JAMA Cardiol*. 2022;7(5):484–491. doi:10.1001/jamacardio.2022.0117
2. Motawea KR, Gaber H, Singh RB, et al. Effect of early metoprolol before PCI in ST-segment elevation myocardial infarction on infarct size and left ventricular ejection fraction. A systematic review and meta-analysis of clinical trials. *Clin Cardiol*. 2022;45(10):1011–1028. doi:10.1002/clc.23894
3. Zhang H, Qiu B, Zhang Y, et al. The value of pre-infarction angina and plasma D-dimer in predicting no-reflow after primary percutaneous coronary intervention in ST-segment elevation acute myocardial infarction patients. *Med Sci Monit*. 2018;24:4528–4535. doi:10.12659/msm.909360
4. Wang X, Wei L, Wu Y, et al. ST-segment elevation predicts the occurrence of malignant ventricular arrhythmia events in patients with acute ST-segment elevation myocardial infarction. *BMC Cardiovasc Disord*. 2023;23(1):61. doi:10.1186/s12872-023-03099-w
5. Deo R, Albert CM. Epidemiology and genetics of sudden cardiac death. *Circulation*. 2012;125(4):620–637. doi:10.1161/circulationaha.111.023838
6. Mehta RH, Starr AZ, Lopes RD, et al. Incidence of and outcomes associated with ventricular tachycardia or fibrillation in patients undergoing primary percutaneous coronary intervention. *JAMA*. 2009;301(17):1779–1789. doi:10.1001/jama.2009.600
7. Carmeliet E. Cardiac ionic currents and acute ischemia: from channels to arrhythmias. *Physiol Rev*. 1999;79(3):917–1017. doi:10.1152/physrev.1999.79.3.917
8. Hundahl LA, Tfelt-Hansen J, Jespersen T. Rat models of ventricular fibrillation following acute myocardial infarction. *J Cardiovasc Pharmacol Ther*. 2017;22(6):514–528. doi:10.1177/1074248417702894
9. Gheeraert PJ, De Buyzere ML, Taeymans YM, et al. Risk factors for primary ventricular fibrillation during acute myocardial infarction: a systematic review and meta-analysis. *Eur Heart J*. 2006;27(21):2499–2510. doi:10.1093/eurheartj/ehl218
10. Jabbari R, Engstrøm T, Glinge C, et al. Incidence and risk factors of ventricular fibrillation before primary angioplasty in patients with first ST-elevation myocardial infarction: a nationwide study in Denmark. *J Am Heart Assoc*. 2015;4(1):e001399. doi:10.1161/jaha.114.001399
11. Huang J, Peng X, Fang Z, et al. Risk assessment model for predicting ventricular tachycardia or ventricular fibrillation in ST-segment elevation myocardial infarction patients who received primary percutaneous coronary intervention. *Medicine*. 2019;98(4):e14174. doi:10.1097/md.0000000000014174
12. Grubb BP, Durzinsky D, Mancini MC, et al. Serum creatine kinase activity and sensing characteristics after intraoperative arrhythmia induction using implantable defibrillator rate sensing leads. *Pacing Clin Electrophysiol*. 1992;15(1):9–13. doi:10.1111/j.1540-8159.1992.tb02894.x
13. Rajan GP, Zellweger R. Cardiac troponin I as a predictor of arrhythmia and ventricular dysfunction in trauma patients with myocardial contusion. *J Trauma*. 2004;57(4):801–808. doi:10.1097/01.ta.0000135157.93649.72
14. Hosseinpoor Z, Farzanegan B, Seyyedi SR, et al. Drug interactions and creatinine levels are associated with QTc prolongation in intensive care units: a prospective, observational study. *Drug Metab Pers Ther*. 2019;34(4). doi:10.1515/dmpt-2019-0022
15. Charnock JS. Lipids and cardiac arrhythmia. *Prog Lipid Res*. 1994;33(4):355–385. doi:10.1016/0163-7827(94)90023-x
16. Hohmann C, Pfister R, Mollenhauer M, et al. Inflammatory cell infiltration in left atrial appendageal tissues of patients with atrial fibrillation and sinus rhythm. *Sci Rep*. 2020;10(1):1685. doi:10.1038/s41598-020-58797-8
17. Smorodinova N, Bláha M, Melenovský V, et al. Analysis of immune cell populations in atrial myocardium of patients with atrial fibrillation or sinus rhythm. *PLoS One*. 2017;12(2):e0172691. doi:10.1371/journal.pone.0172691
18. Yamashita T, Sekiguchi A, Iwasaki YK, et al. Recruitment of immune cells across atrial endocardium in human atrial fibrillation. *Circ J*. 2010;74(2):262–270. doi:10.1253/circj.cj-09-0644
19. Moons KG, Altman DG, Reitsma JB, et al. Transparent Reporting of a Multivariable Prediction Model for Individual Prognosis or Diagnosis (TRIPOD): explanation and elaboration. *Ann Internal Med*. 2015;162(1):W1–73. doi:10.7326/m14-0698
20. Battersby NJ, Bouliotis G, Emmertsen KJ, et al. Development and external validation of a nomogram and online tool to predict bowel dysfunction following restorative rectal cancer resection: the POLARS score. *Gut*. 2018;67(4):688–696. doi:10.1136/gutjnl-2016-312695
21. Krumholz HM, Normand SL, Wang Y. Trends in hospitalizations and outcomes for acute cardiovascular disease and stroke, 1999–2011. *Circulation*. 2014;130(12):966–975. doi:10.1161/circulationaha.113.007787
22. Killip T 3rd., Kimball JT. Treatment of myocardial infarction in a coronary care unit. A two year experience with 250 patients. *Am J Cardiol*. 1967;20(4):457–464. doi:10.1016/0002-9149(67)90023-9
23. Del Buono MG, Montone RA, Rinaldi R, et al. Clinical predictors and prognostic role of high Killip class in patients with a first episode of anterior ST-segment elevation acute myocardial infarction. *J Cardiovasc Med*. 2021;22(7):530–538. doi:10.2459/JCM.0000000000001168
24. Khot UN, Jia G, Moliterno DJ, et al. Prognostic importance of physical examination for heart failure in non-ST-elevation acute coronary syndromes: the enduring value of Killip classification. *JAMA*. 2003;290(16):2174–2181. doi:10.1001/jama.290.16.2174
25. Francis Stuart SD, De Jesus NM, Lindsey ML, et al. The crossroads of inflammation, fibrosis, and arrhythmia following myocardial infarction. *J Mol Cell Cardiol*. 2016;91:114–122. doi:10.1016/j.yjmcc.2015.12.024
26. Nienhuis MB, Ottervanger JP, de Boer MJ, et al. Prognostic importance of creatine kinase and creatine kinase-MB after primary percutaneous coronary intervention for ST-elevation myocardial infarction. *Am Heart J*. 2008;155(4):673–679. doi:10.1016/j.ahj.2007.11.004
27. Macdonald JE, Struthers AD. What is the optimal serum potassium level in cardiovascular patients? *J Am Coll Cardiol*. 2004;43(2):155–161. doi:10.1016/j.jacc.2003.06.021
28. Robinson VM, Alsalahat I, Freeman S, et al. A carvedilol analogue, VK-II-86, prevents hypokalaemia-induced ventricular arrhythmia through novel multi-channel effects. *Br J Pharmacol*. 2022;179(11):2713–2732. doi:10.1111/bph.15775
29. Liu P, Yang Y, Zhang H, et al. The effect of TWIK-1 two pore potassium channels on cardiomyocytes in low extracellular potassium conditions. *Front Biosci*. 2023;28(3):51. doi:10.31083/j.fbl2803051
30. Yamada S, Inaba M. Potassium metabolism and management in patients with CKD. *Nutrients*. 2021;13(6):1751. doi:10.3390/nu13061751
31. Mahalle N, Garg M, Kulkarni M, et al. Association of inflammatory cytokines with traditional and nontraditional cardiovascular risk factors in Indians with known coronary artery disease. *Ann Med Health Sci Res*. 2014;4(5):706–712. doi:10.4103/2141-9248.141525

32. Lim J, Davies A, Brienesse S, et al. Inflammatory cell response following ST-elevation myocardial infarction treated with primary percutaneous coronary intervention and its impact on cardiovascular outcomes: a systematic review and meta-analysis. *Int J Cardiol*. 2023;376:1–10. doi:10.1016/j.ijcard.2023.01.082
33. Su M, Ouyang X, Song Y. Neutrophil to lymphocyte ratio, platelet to lymphocyte ratio, and monocyte to lymphocyte ratio in depression: a meta-analysis. *J Affect Disord*. 2022;308:375–383. doi:10.1016/j.jad.2022.04.038
34. Zhang S, Diao J, Qi C, et al. Predictive value of neutrophil to lymphocyte ratio in patients with acute ST segment elevation myocardial infarction after percutaneous coronary intervention: a meta-analysis. *BMC Musculoskelet Disord*. 2018;18(1):75. doi:10.1186/s12872-018-0812-6
35. Fagundes A Jr., Ruff CT, Morrow DA, et al. Neutrophil-lymphocyte ratio and clinical outcomes in 19,697 patients with atrial fibrillation: analyses from ENGAGE AF- TIMI 48 trial. *Int J Cardiol*. 2023;386:118–124. doi:10.1016/j.ijcard.2023.05.031
36. van der Laan AM, Hirsch A, Robbers LF, et al. A proinflammatory monocyte response is associated with myocardial injury and impaired functional outcome in patients with ST-segment elevation myocardial infarction: monocytes and myocardial infarction. *Am Heart J*. 2012;163(1):57–65.e52. doi:10.1016/j.ahj.2011.09.002
37. Tsujioka H, Imanishi T, Ikejima H, et al. Impact of heterogeneity of human peripheral blood monocyte subsets on myocardial salvage in patients with primary acute myocardial infarction. *J Am Coll Cardiol*. 2009;54(2):130–138. doi:10.1016/j.jacc.2009.04.021
38. Zheng X, Huang R, Liu G, et al. Development and verification of a predictive nomogram to evaluate the risk of complicating ventricular tachyarrhythmia after acute myocardial infarction during hospitalization: a retrospective analysis. *Am J Emerg Med*. 2021;46:462–468. doi:10.1016/j.ajem.2020.10.052